Table I. *Reactivity of Malaria Immune Sera with Trypsin-sensitive and Trypsin-resistant Antigens on the pRBC Surface*

	Agglutination* of trypsin-digested pRBCs‡ (μg/ml trypsin)			
Sera	0	1	10	100
011K	4+	4+	3+	1+
022L	1+	−	−	−
080K	4+	4+	−	−
118K	1+	1+	1+	−
119L	4+	3+	−	−
142L	2+	1+	1+	1+
163L	4+	2+	2+	2+
174L	2+	1+	1+	−
241L	−	−	−	−
Antigen	Amount detected on pRBC surface§			
	%			
PfEMP1	100	0	0	0
39-kD rifin	100	97	71	65
35-kD rifin	100	74	37	15

*Agglutination of trophozoite-containing pRBCs after incubation for 1 h at 37°C with 1:2 dilution of sera from donors from Kenya (nos. 11, 80, and 118) or Liberia (nos. 22, 119, 142, 163, 174, and 241). Agglutination was graded as described in Materials and Methods.
‡FCR3S1 pRBCs were incubated with enzyme at 37°C for 10 min.
§The amount of radioiodinated antigen was estimated by PhosphorImager-assisted quantitation of cpm in the corresponding SDS-PAGE bands.

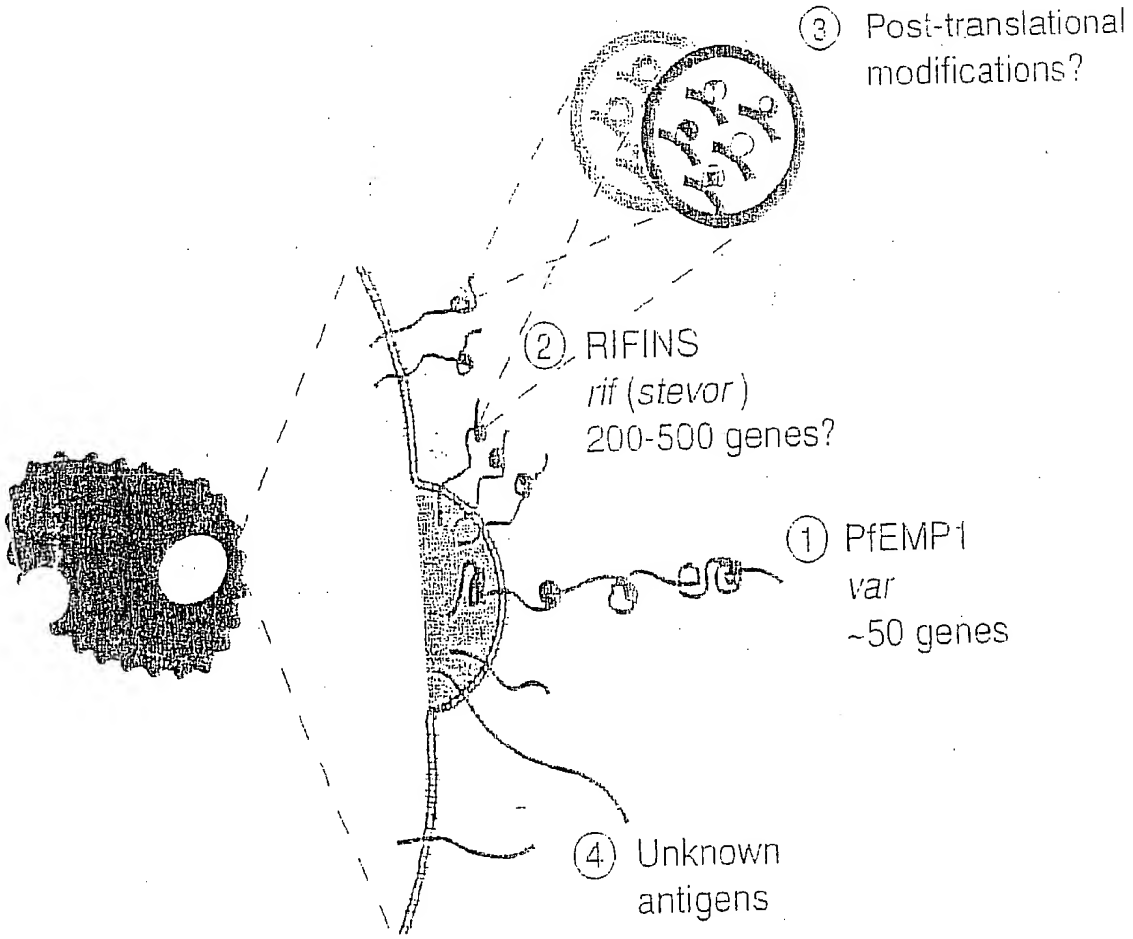

Figure 7. Antigenic diversity on the infected erythrocyte surface. Sources of the structural variation created by *P. falciparum* on its host cell can be: (1) the large variant antigen PfEMP1, encoded by the gene family *var*, with ~50 genes per genome; (2) the small antigens rifins encoded by the multigene family *rif*, also termed *stevor* (200–500 genes) (the location of these products at knobs is not presently established); (3) modifications (not yet defined) of the translated polypeptide resulting in further layers of molecular microheterogeneity; or (4) additional parasite antigens transported to the pRBC surface.